Anti-HVEM [HMHV-1B18] Standard Size Ab01026-2.3

This antibody was created using our proprietary Fc Silent™ engineered Fc domain containing key point mutations that abrogate binding to Fc gamma receptors.

This is a reformatted Fc Silent™ chimeric antibody made using the variable domain sequences of the original hamster (Armenian) antibody format to improve compatibility with existing reagents, assays, and techniques.

Isotype and Format: Mouse IgG2a, Fc Silent™, Kappa
Clone Number: HMHV-1B18
Alternative Name(s) of Target: CD270; TNF Receptor-like 2; ATAR; Herpes virus Entry Mediator A; HVEA; Herpes virus Entry Mediator; LIGHTR; Tumor Necrosis Factor Receptor-Like Gene2; Tumor Necrosis Factor Receptor-Like 2; TR2; Tumor Necrosis Factor Receptor Superfamily Member 14; TNFRSF14; CD40-Like Protein
UniProt Accession Number of Target Protein: Q71F55
Published Application(s): Blocking, functional assays, IP, WB, FC
Published Species Reactivity: Mouse
Immunogen: This antibody was raised by immunising Armenian hamsters with mouse HVEM:Fc fusion protein.
Specificity: This antibody is specific for Herpes Virus Entry Mediator (HVEM, TR2), a type I transmembrane protein of TNF-receptor superfamily. This receptor, which is expressed on most cell types, including T cells, B cells, monocytes, neutrophils, and dendritic cells. Binding of HSV viral envelope glycoprotein D (gD) to this receptor protein has been shown to be part of the viral entry mechanism. The cytoplasmic region of HVEM was found to bind to several TRAF family members, which may mediate the signal transduction pathways that activate the immune response. HVEM has also been demonstrated to be a unique ligand for BTLA (B and T lymphocyte attenuator). The conservation of the BTLA-HVEM interaction between mouse and human suggests that this system is an important pathway regulating lymphocyte activation and/or homeostasis in the immune response.
Application Notes: This antibody has been used in FACS to demonstrate that lymphatic endothelial cells mediate deletion only via programmed cell death-1 (PD-1) ligand 1 (Tewalt et al 2012) and in Western Blot to study the role of LIGHT in the pathogenesis of hepatitis (Anand et al 2006). This antibody has been also been used in vivo experiments to study the mechanisms by which TNFSF14 functions to promote airway remodelling in asthma (Sibilano et al 2016), to confirm that costimulatory role through HVEM is not

necessary for LIGHT-mediated liver inflammation (Anand et at 2006), and to investigate the role that herpesvirus entry mediator plays in the development of experimental conjunctivitis (Ishida et al, 2012). Treatment with this antibody has been observed to diminish plasma levels of antigen-specific IgG1 and IgE antibodies in mouse asthma models (Sibilano et al 2016), to interfere with the LIGHT-HVEM interaction but not interaction between B and T lymphocyte attenuator (BTLA) and HVEM in mouse hepatitis models (Anand et at 2006), and NOT to affect the development of experimental conjunctivitis in either the induction or the effector phase (Ishida et al, 2012).

Antibody First Published in: Waka Ishida et al. B and T lymphocyte attenuator regulates the development of antigen-induced experimental conjunctivitis. Graefes Arch Clin Exp Ophthalmol. 2012 Feb;250(2):289-95. PMID:21779950

Note on publication: Describe the use of this antibody, together with the anti-BTLA antibody, to investigate the roles that B and T lymphocyte attenuator (BTLA) and herpesvirus entry mediator (HVEM) play in the development of antigen-induced experimental conjunctivitis (EC).

Product Form

Size: 200 µg Purified antibody.
Purification: Protein A affinity purified
Supplied In: PBS with 0.02% Proclin 300.
Storage Recommendation: Store at 4ºC for up to 3 months. For longer storage, aliquot and store at -20ºC.
Concentration: 1 mg/ml.

Important note – This product is for research use only. It is not intended for use in therapeutic or diagnostic procedures for humans or animals.